II. Coronary Heart Disease in the Framingham Study

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A continuing epidemiological study of heart disease was established in Framingham, Mass., during the period of 1948–1950. This study is concerned with measurements of the extent and development of cardiovascular disease in a cross-section of the population aged 30–59 on January 1, 1950, and with the study of those environmental and personal factors which are associated with the subsequent appearance and progression of cardiovascular diseases.[1] Data are obtained primarily from biennial examinations conducted in a clinic maintained especially for this study. It is planned to continue observation of the group of subjects for 20 years. The present report describes the experience with arteriosclerotic heart disease observed in the four years following each individual's initial examination.

Methods

A description of the town of Framingham and the methods by which the sample was drawn have been reported.[1] The response to this sample invitation was also discussed.[2] A summary of the cooperation obtained from the original sample in the first examination is shown in Table 1. The original health status and subsequent course of the persons who were selected, but not examined at the first examination, is the subject of continuing investigation, since this information has an important bearing on the generality of the findings of the study.

The original plan of the Framingham study provided for following only the persons in the sample who were free of arteriosclerotic heart diseast (ASHD) and hypertensive cardiovascular disease (HCVD). It was soon apparent that restriction of attention to this group, identified with difficulty at best, would result in the loss of much useful information concerning the life history of cardiovascular disease. Furthermore, it appeared that exclusion of diseased persons after the first examination would have weakened the clinic's relation with the community. For these reasons, persons with diagnoses of cardiovascular disease have been followed in this study along with those found free of this disease.

Table 1—Response to the Initial Examination

Response Status	No. of Persons	Per cent
Total sample	6,510	100
Examined	4,469	68.6
Not examined	2,041	31.4
Moved out of Framingham	421	6.5
Died before initial examination	74	1.1
Ill or incapacitated	74	1.1
Noncooperative	1,472	22.6

The organization of the clinic where the subjects were examined, the content of the examination, and the laboratory methods have also been described.[1] A detailed medical history and a physical examination was carried out by a physician. The findings of these procedures were entered on standard forms. Each

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subject was seen by a second physician who evaluated the findings of the first. The laboratory examinations included measurements of vital capacity, height, and weight; an electrocardiogram; a standard chest film; measurement of hemoglobin, glucose, uric acid, cholesterol, and phospholipid; and serological test for syphilis. The protein, reducing material, and specific gravity were measured in the urine. Other procedures, such as 70 mm photofluorogram, electrokymography, ballistocardiography, and ultracentrifugal analysis of serum lipoproteins have been made at one or another of the successive examinations, but these have not been used for diagnostic classification. The chemical studies will be described in detail in another report.

At the completion of the examination the findings were briefly discussed with the subject and he was advised that he and the physician he had named would soon receive a report of the results of the procedure. The medical staff tried to avoid contributing medical advice or therapy beyond that necessary in situations of medical emergencies, e.g., breast tumors, signs of congestive failure, untreated diabetes, or unrecognized and acute evidence of coronary heart disease. The report sent the physician was analogous to a hospital discharge summary and listed the diagnoses established, but it did not contain recommendations for therapy. The research records were available to these physicians upon request. The subject was sent a form letter which said in effect, and as appropriate, "you were found to be free of cardiovascular disease," "we have confirmed the existence of a condition of which you were aware," or "we believe you should see your doctor concerning our findings." While it would be desirable from an epidemiological point of view to observe the occurrence of disease among individuals who were unaffected by knowledge of the observations this is not feasible, because the clinic physicians were obligated in some instances, by the urgency of the findings, to encourage the subject to place himself under the care of his physician.

Follow-Up Procedure

The subjects were examined with the same procedures on or near the biennial anniversary of their original examination. They were reached by telephone or mail and invited to accept an appointment. If a subject did not accept, the reason for the refusal was sought and recorded. The health status of each person who could not be reached directly was sought through relatives, neighbors, or associates. Every effort was made to determine whether the subject was living or dead. In this way the optimal information of an examination in the clinic was supplemented with less reliable information obtained through the subject's personal associates.

An analysis of the follow-up status of the 4,469 subjects who were initially examined is shown in Table 2. It is instructive to measure this follow-up performance for two kinds of sources of information, namely, "clinical" data for living subjects and "mortality" data for the others. This procedure is appropriate, since these sources are often of quite different reliability. The clinical information was obtained largely from the observation of subjects at their successive visits to the clinic, or from the records of nearby hospitals. The death data arose from the files of medical examiners, state and local health departments, and the clinical or autopsy reports of hospital deaths. During the early years of the study there were two general hospitals in the community. All the admissions of the subjects in the study to the larger (and presently the only) hospital in Framingham were reviewed in order to obtain data for those admitted for cardiovascular diseases.

Table 2—Completeness of Clinical and Mortality Follow-Up Four Years After Initial Examination

	No. of Persons	Per cent
Clinical Follow-Up		
Total sample	4,469	100
Persons with complete 4-year follow-up	3,984	89.1
Received examination III *	3,931	
Skipped examination III, but examined at IV	53	
Persons with 2-year clinical follow-up	193	4.3
Refused examination at III	113	
Moved before examination III	45	
Died before examination III	35	
No clinical follow-up	292	6.5
Refused examination at II and III	194	
Moved before examination II	60	
Died before examination II	38	
Mortality Follow-Up		
Total sample	4,469	100
Known dead in 4-year follow-up period	73	1.6
Known living at end of 4-year follow-up	4,349	97.3
Known living at 2 years, status unknown at 4 years	19	0.4
Follow-up status unknown	28	0.6

* The successive biennial examinations of the Framingham study are designated with Roman numerals, thus Examination III was 4 years after Examination I.

This information supplemented the history given by subjects who returned to the research clinic for examination, and it served as a reliable source of information on hospitalized disease developing in persons who did not return to the research clinic. Information concerning admissions to the second hospital, as for other surrounding hospitals, was not obtained for all sample persons in a systematic way, but was requested when the subject returned to the clinic and indicated that he had been admitted to a hospital with a disease known or suspected to be cardiovascular.

The data of Table 2 indicate that 89 per cent of persons initially examined who could have been followed were followed clinically for four years. An additional 4 per cent of the population were followed for two years. In terms of man-years of exposure, clinical data are available for 91 per cent of the years lived in the four-year, follow-up period. Mortality follow-up was much more complete. Only 1 per cent of the total sample could not be accounted for in respect to mortality status at the end of the four-year period and many persons in this group had been followed for two years.

Classification of Arteriosclerotic Heart Disease (ASHD)

The criteria used for the classification of arteriosclerotic heart disease followed those of the New York Association.[3] For the present purposes the following manifestations of ASHD are considered:

1. Myocardial infarction, with either or both historical and laboratory evidence that was suf-

ficient for all observers to make this diagnosis.

2. Angina pectoris, with sufficient symptoms to lead all observers to agree with this diagnosis.

3. Coronary occlusion. "Sudden death" attributed to coronary heart disease.

4. Myocardial fibrosis. Death, with either clinical evidence of progressive cardiac failure in the absence of apparent cause, or with autopsy evidence of myocardial fibrosis which could be attributed to atherosclerosis of the coronary arteries.

5. Myocardial infarction, possible, ECG evidence only. Persons without a history consistent with myocardial infarction, but presenting electrocardiographic evidence suggesting, but not conclusive of, myocardial infarction.*

"Arteriosclerosis of the aorta" is not included in the present consideration and there were no instances of "sclerosis of a valve," each of which is an anatomical diagnosis. The decision to include the subjects with "myocardial infarction, possible, ECG evidence only" was based upon the demonstration that the subsequent clinical courses of persons so classified at the initial examination were more often complicated by definite ASHD than were those of persons classified as "possible myocardial infarction by history" alone. The decision was also influenced by the greater objectivity of ECG evidence.

There were many subjects with questionable evidence of coronary heart disease including: (1) those subjects from whom a history suggestive of myocardial infarction was obtained, but without any supporting laboratory or clinical evidence, and (2) those subjects from whom a history suggestive of angina pectoris was obtained, in which the two observers were either in disagreement, or one or both were uncertain concerning the interpretation of these symptoms. These questionable manifestations of disease were grouped with other abnormalities, perhaps attributable to arteriosclerosis, such as generalized cardiac enlargement by x-ray, and left ventricular hypertrophy by ECG, or nonspecific abnormal T waves, each occurring in the absence of known causation. None of these is included in the category "arteriosclerotic heart disease" in this analysis. The relegation of these uncertain diagnoses to a less conspicuous category in the present analysis has an important influence upon the prevalence and incidence rates of ASHD. It seems clear that the final disposition of persons with these diagnoses of possible ASHD in a classification system cannot be solved until a long-term observation of their clinical implications has been made. Such an objective is a major concern of the Framingham study.

It should be understood that those persons with questionable evidence of ASHD at the first examination were, for purposes of incidence measurements, placed with all the other persons in the population. Neither those, nor many others in the population can be considered strictly normal people for, in addition to those with a question of ASHD, there were others with rheumatic heart disease (RHD), hypertensive cardiovascular disease (HCVD), and other diseases. These incidence estimates for a four-year period represent the rates of appearance of diagnosable ASHD in a population from which only those persons with definite ASHD have been excluded.

The classification of disease in subjects who died during the follow-up interval presents a different problem. The best available evidence concerning these subjects is derived from a hospital record of the terminal illness which may include an autopsy report. More often the information was only that included in a report of the medical examiner or

* The abnormalities observed were generally patterns of deep Q_3 and Q_{AVF} with or without inverted T wave which were considered to represent possible posterior infarction, and deep Q_1 with inverted T_1, usually with inverted T wave, in $V_{4,5,6}$, and with absent R waves over the right ventricle. The latter changes were consistent with a diagnosis of possible anterior infarction.

Table 3—Prevalence of Arteriosclerotic Heart Disease at the Initial Examination, by Age and Sex

Age	Males			Females		
	No. Examined	No. with ASHD	Rate/1,000	No. Examined	No. with ASHD	Rate/1,000
Total	2,024	48	24	2,445	28	11
30–44 *	1,083	5	5	1,317	7	5
30–34 *	335	2		389	1	
35–39	390	2		491	1	
40–44	358	1		437	5	
45–62	941	43	46	1,128	21	19
45–49	313	5		379	3	
50–54	319	18		365	8	
55–59	236	15		321	8	
60–62	73	5		63	2	

* Includes 5 males and 9 females aged 29 years; there was no ASHD among these people.

in the death certificate. Since ASHD is often a fatal disease, and particularly apt to end with "sudden death," the accurate etiological classification of these subjects constitutes one of the serious limitations of this and all similar studies of ASHD. The duration of the terminal illness is of critical importance in determining the extent of clinical evaluation and thus documentation of cause of death. The deaths which occurred in the sample population without sufficient clinical and laboratory evidence to permit a diagnosis of myocardial infarction, but which by their suddenness, the immediate appearance of the patient, and the exclusion of other probable causes lead the officials to conclude that this was caused by ASHD, have been called "coronary occlusion without myocardial infarction" in the present evaluation.

The tabulations shown represent an unduplicated count of persons. This was accomplished for subjects with more than one manifestation of ASHD by assigning a priority of importance to the various diagnostic categories. In diminishing rank this priority was

1. Myocardial infarction
2. Coronary occlusion without myocardial infarction
3. Angina pectoris
4. Myocardial infarction, possible, ECG evidence only
5. Myocardial fibrosis

In practice the application of this priority rule was not often necessary. One man who died suddenly has been classified as coronary occlusion, although he was known to have had angina pectoris. There were three persons classified as angina pectoris who also showed possible myocardial infarction by electrocardiographic evidence.

Results

Prevalence of Arteriosclerotic Heart Disease in 1949–1952 *

One product of the initial examination of the subjects in the sample population was an estimate of the prevalence of ASHD in the sample examined. These data are presented by sex and five-year

* Completion of the first cycle of examinations in the Framingham study required about four years.

Table 4—Number of Subjects by Type of Arteriosclerotic Heart Disease Diagnosed at Initial Examination, by Age and Sex

Diagnosis	Males			Females		
	Total	30–44	45–62	Total	30–44	45–62
Total	48	5	43	28	7	21
Myocardial infarction	17	2	15	1	–	1
Angina pectoris	20	2	18	18	5	13
Possible myocardial infarction, ECG evidence only	11	1	10	9	2	7

age interval in Table 3 for all diagnostic categories of ASHD combined. When the diagnoses are considered collectively the prevalence rates for the two sexes are very similar through age 49. In the sixth decade of life a remarkable increase in prevalence occurs in both sexes, but the increase is larger for men. The numbers of subjects over 59 years were too small to permit generalization about trend. It should be emphasized that these prevalence rates are based on counts of survivors of ASHD, as defined, who were able to attend the clinic, and the rates are, therefore, minimum estimates of the true prevalence.

In Table 4 these prevalence data are arranged by diagnostic category as well as by age and sex. Only the actual numbers of persons are shown, and thus the comparisons between diagnoses should be made only for specific age and sex groups. The most notable fact is the predominance of the diagnosis of angina pectoris in women in proportion to the total number of women with ASHD. Conversely, myocardial infarction was an uncommon disease in these women.

Table 5—Four-Year Incidence of Arteriosclerotic Heart Disease Among Persons Free of This Disease at Initial Examination, by Age and Sex

Age at Initial Examination	Males			Females		
	Population at Risk	New Disease	Rate/1,000	Population at Risk	New Disease	Rate/1,000
Total	1,976	65	33	2,417	32	13
30–44 *	1,078	13	12	1,310	–	–
30–34 *	333	3		388	–	
35–39	388	5		490	–	
40–44	357	5		432	–	
45–62	898	52	58	1,107	32	29
45–49	308	8		376	8	
50–54	301	15		357	8	
55–59	221	23		313	14	
60–62	68	6		61	2	

* Includes 5 males and 9 females aged 29 years; there was no new ASHD among these people.

Incidence of New ASHD in the Four-Year Follow-Up Interval

The four-year incidence of ASHD defined above as myocardial infarction, coronary occlusion, angina pectoris, myocardial fibrosis and possible myocardial infarction by ECG is shown by sex and age in Table 5. It should be emphasized again that the population at risk for this observation was composed of all the subjects in the sample except those with the diagnosis of ASHD at entry to the study as shown in Tables 3 and 4. The incidence rates were highly dependent on age and they were two to three times higher in males than in females. There is a suggestion that an upturn in the trend of incidence rates for each sex occurs at about age 45, a conclusion which has been used in grouping these data for subsequent analysis.

The incidence data are arranged by diagnosis for specific age and sex groups in Table 6. Since both fatalities and surviving persons are counted, it is of interest to consider the early mortality characteristics of ASHD. The prominence of "sudden death" as a manifestation of ASHD in men is a striking feature of these observations. Thirteen of the 43 men with "heart attacks" (i.e., myocardial infarction or coronary occlusion) were classified as "sudden death"; these persons, by the nature of the event, would not have reached the care and study of physicians. (This proportion for men aged 45–62 was 11 out of 31.) These data imply that studies of ASHD which are based upon autopsy material or hospital discharges are only partial observations of the disease. Angina pectoris, while almost as frequent a new manifestation of ASHD as myocardial infarction for men of all ages, occurred more commonly after age 45 than before. The data in Table 6 have been grouped into two age groups, 30–44 and 45–62 years. The older age group includes 80 per cent of all ASHD incidence for men and the entirety of the ASHD incidence in women.

The phenomenon of sudden death was the only manifestation of disease in almost half of all subjects with myocardial infarction who developed new ASHD exclusive of angina pectoris and possible myocardial infarction by ECG. In contrast to the sudden deaths are the so-called "silent" infarctions, the surviving subjects for whom the history of symptoms is absent or insufficient to contrib-

Table 6—Number of Subjects Developing New Disease in a Four-Year Period by Diagnosis and by Age and Sex

Diagnosis	Males			Females		
	Total	30–44	45–62	Total	30–44	45–62
Total	65	13	52	32	—	32
Myocardial infarction	30	10	20	4	—	4
Surviving persons *	27	9	18	3		3
Fatalities	3	1	2	1		1
Coronary occlusion w/o infarction †	13	2	11	1	—	1
Myocardial fibrosis with death	2	—	2	1	—	1
Angina pectoris	20	1	19	24	—	24
Possible myocardial infarction	—	—	—	2	—	2

* Survived to fourth anniversary of initial examination except for two persons who died from second coronary occlusion before Examination III.

† "Sudden death."

ute to the diagnosis, who were diagnosed on the basis of ECG evidence only. Seven of these "silent" infarctions occurred during the four-year follow-up period covered by this report, four among men and three among women. Indeed, one of these asymptomatic heart attacks was occurring (but was not diagnosed) at the time of the initial examination, since a second ECG made at a special examination one month later confirmed the usual evolution of ECG changes characteristic of a myocardial infarction. The total of seven of these asymptomatic or "silent" coronary attacks which occurred in the four-year follow-up period is to be compared with the total incidence of 36 persons with definite or possible myocardial infarction. The subject of "silent" infarctions has been discussed recently by Snow, Jones, and Daber [4] who concluded that the wide range of frequencies of this form of coronary disease was the result of the variable criteria applied in different studies. It is apparent that the "silent" coronary is an important part of the disease. Furthermore, there were persons in this study group who at some time showed definitive ECG evidence of a myocardial infarction but who later lost all residual evidence of ECG changes. The difficulty of diagnosing ASHD, underestimated by some,[5] will no doubt continue to obscure the evaluation of causal relationships.

Angina pectoris represents about one-third of all the new ASHD in men and this disease generally appeared in men after age 45. The new ASHD in women, occurring always after age 45, was predominantly angina pectoris. These data emphasize the dissimilarity of ASHD in the two sexes. The predominance of angina pectoris as the manifestation of ASHD in women suggests several possible explanations, none of which can be established with these data: (1) the diagnosis may be faulty. The greater prevalence of neurocirculatory asthenia in females [6] may lead to confusion with this disease, and (2) the manifestations of ASHD in women may differ from those in men. These problems may be resolved with additional observation. The tendency of the diagnosis of definite but uncomplicated angina pectoris in women to shift to questionable angina pectoris in successive examinations, and perhaps back to definite angina pectoris, suggests that the clinical interpretation of symptoms of angina pectoris in women is less reliable than in men. This shifting diagnosis was found despite rigorous attention to questioning technic and interpretative criteria. The variability must include, among other things, diagnostic error and the disappearance of symptoms of angina pectoris as a result of a decrease of physical activity or an adaptation to the symptoms. It will be necessary to trace the outcome of angina pectoris, the common manifestation of ASHD in women, in order to determine whether this condition is an antecedent stage of myocardial infarction and coronary occlusion, or whether the disease takes a different course in women.

The Association of Certain Clinical Attributes with the Development of ASHD

The Framingham study is predicated on the belief that an understanding of the pathogenesis of ASHD will be facilitated by a careful recording of the characteristics of the subjects before disease develops and analysis of antecedent differences between the group which develops disease while under observation and the group which does not. For the present analysis only the male subjects in the age range 45–62 at initial examination have been considered, since this is the only category in which new disease occurred with sufficient frequency in the first four years of follow-up to permit detailed analysis.

There were 52 men in this age group, free of disease at the initial examination, who developed ASHD in the subsequent four years; these represent 80 per cent of all men in the study who developed new ASHD in this follow-up period. The restriction of this analysis to the 45–62 age group was also influenced by the recognition that ASHD occurring before that age is relatively uncommon and possibly a different disease, since it is generally characterized by a more profound disturbance of cholesterol metabolism.[7, 8]

The analytical procedure in the following sections consists of the classification of individuals by various characteristics, such as blood pressure level, relative weight, or serum cholesterol level as these were observed at the initial examination. For each of these subgroupings of individuals who were at risk of coronary disease for the four-year follow-up, there is recorded the number of individuals who developed new disease and the incidence rate per 1,000 for the four-year period. It is then possible to test the null hypothesis that the observed differences between subgroupings are no greater than would be expected by chance variation of an incidence rate common to all subgroupings, given the numbers under observation. In the sections immediately following are shown data for blood pressure and hypertensive disease, relative fatness, and serum cholesterol which suggest that for each of these characteristics taken individually the assumption of no association appears untenable. In a subsequent section it will be shown that the three factors taken together will permit even stronger inferences as to association of these factors and risk of ASHD.

High Blood Pressure

It is widely believed that high blood pressure (HBP) augments the develop-

Table 7—Hypertension and Hypertensive Heart Disease in Relation to Incidence of ASHD in Four-Year Follow-Up, Males 45–62

	Population at Risk	New Disease	Rate/1,000
All persons	898	52	58
Definite hypertensive heart disease	82	8	98
Definite hypertension	124	10	81
Possible hypertensive heart disease	92	7	76
Borderline hypertension without heart disease	243	15	62
Normotension	310	8	26
(Cerebrovascular accident, or rheumatic heart disease not included above)	(47)	(4)	

Normotension: Left arm blood pressure determinations are below 140/90 on independent observations by two physicians.
Hypertension: Definite hypertension was diagnosed when both left arm systolic blood pressure determinations recorded by physicians were 160 mm Hg or over and/or all left arm diastolic blood pressures were 95 or over, or when each blood pressure reading was either a high systolic (160 or over) or a high diastolic (95 or over) level.
Borderline hypertension: All others.
Hypertensive heart disease: When in addition to definite hypertension, definite left ventricular hypertrophy or generalized cardiac enlargement by x-ray or left ventricular hypertrophy by ECG was present.
Possible hypertensive heart disease: When hypertension was definite in the presence of doubtful left ventricular hypertrophy (generalized cardiac enlargement) by x-ray or ECG or hypertension was doubtful, but with definite left ventricular hypertrophy or generalized cardiac enlargement or both hypertension and left ventricular hypertrophy (or generalized cardiac enlargement) were doubtful.

Table 8—Certain X-Ray and Electrocardiographic Findings in Relation to Incidence of ASHD in Four-Year Follow-Up, Males 45–62

	Population at Risk	New Disease	Rate/1,000
All persons	898	52	58
LVH or GCE by chest x-ray *	193	10	52
Definite	163	10	61
Possible	30	—	—
LVH or LV strain by electrocardiogram *	59	12	203
Definite	29	7	†
Possible	30	5	†

* These categories are not mutually exclusive.
† Rates not computed for base less than 50.

ment of ASHD. The prominence of this relationship has led some clinicians to conclude that the major part of the hypertension problem lies in the prevention of atherosclerosis. The relation of high blood pressure and hypertensive heart disease to the four-year incidence of ASHD in the Framingham study are shown in Table 7. The incidence rates indicate a trend from the low of 26 per 1,000 for 310 persons with normal blood pressure and free of all other kinds of heart disease to a high of 98 per 1,000 for 82 persons with definite hypertensive heart disease at the initial examination. It is clear that HBP is significantly associated with the incidence of ASHD ($X^2 = 10.2$, $n = 4$, $p = 0.04$).

The relationship of specific signs of hypertensive cardiovascular disease to the incidence of ASHD was considered. In Table 8 the relationship of cardiac hypertrophy measured with the chest film and with the ECG pattern of left ventricular hypertrophy (LVH) is considered. The x-ray diagnosis in this instance is based upon the radiologist's "pattern recognition" of generalized cardiac enlargement (GCE) and left ventricular hypertrophy. While this estimate is highly correlated with frontal area measurements of the heart and thorax which were also made, the latter have not been used in this evaluation. The data in Table 8 indicate that the x-ray diagnoses of definite or possible LVH or GCE were not associated with the development of ASHD. On the other hand, ECG evidence of LVH, whether definite or possible, was highly associated with the incidence of ASHD ($X^2 = 21.7$, $n = 1$, $p < 0.001$). These data raise many questions: Is the effect of HBP mediated through acceleration of atherogenesis, through precipitation of the occlusive event by mural damage which initiates thrombosis, or by myocardial hypertrophy which aggravates the inadequacy of coronary flow? The last proposal is not well supported by the low association of x-ray diagnoses of LVH and GCE with the incidence of new ASHD. The electrocardiographic diagnosis of LVH is, on the other hand, very highly associated with the incidence of new ASHD. It appears that these methods appraise different aspects of cardiac function. The explanation of this difference of association with the appearance of ASHD may lie in a lower sensitivity of this ECG pattern to cardiac damage. It is clear that this finding of LVH by ECG needs further study, because any laboratory measurement

which has predicted a fifth of the men who developed definite ASHD in four years may be adaptable to clinical needs.

Weight *

Since hypertension and obesity have been found to be correlated,[9] it is of interest to examine the relationship of some estimates of fatness and fattening to the incidence of ASHD. This task is complicated by the lack of either precise measurements of fatness or a definitive criterion for judging relative fatness. The actuarial data [10, 11] have tended to show that obesity, especially in men, is associated with a large increase in the risk of ASHD. These conclusions have been based upon very large, if superficially observed, populations with the assessments of obesity derived from weight and height measurements collected by many people under variable conditions. The criteria of obesity in most of those studies have been derived from the actuarial tables of observed weight for height and sex collected in the early part of this century. Because of the limitations of these methods it was elected to establish a reference base of weight by sex and height within the Framingham population.

The present analysis of the relation of weight to ASHD has been based on an index relating height to weight which, in order to prevent confusion with other relative weight indexes, will be termed "Framingham relative weight" (FRW). This index is the ratio (x 100) of an individual's observed weight to the median weight for his sex-height group in the entire Framingham cross-section at the initial examination. The median weights are, thus, based on a population with an age range 29–62 with the distribution of ages shown in Table 3. Medians were computed for the male population for each inch of height in the range 61–73 inches. In this range there was clearly a linear relation between median weight and height and the data were smoothed by use of the following formula (where weight is in pounds and height in inches): Median weight $= 136 + 4$ (ht. -60).

An extrapolation of one inch was made in each direction to cover the height range 60–74 inches which included all but five of the men aged 45 or over. Since obesity is common in this population, it follows that the weight distributions reflect this in the parameters; but the ratio of an individual's observed weight to the median weight for height of the population, which is called the "Framingham relative weight" (FRW), does not presume to judge desirability, only to supply a reasonably stable denominator or reference base for the individual's observed weight.

The data presented in Table 9 support the hypothesis that obesity is related to risk of coronary attack ($X^2 = 12.8$, $n = 3$, $p = 0.005$). The upper tenth of the population in respect to "Framingham relative weight" showed an incidence rate three times that of the population whose weight was below the median for height. There was a regular downward progression of incidence rates from the highest relative weight group down to the median; with the small amount of data at hand there appeared to be no gradient in the group below the median.

The study also provided information on change of weight since age 25 based on the subject's recollection of his weight at that age. Although this kind of information is liable to considerable memory error, it may be a useful alternative way of estimating obesity, if a man's weight at 25 is considered in some sense his "ideal weight." The association of this

* Men were weighed and measured with shirt and shoes removed. Height was recorded to next lower quarter inch and weight to the next lower quarter pound. Tabulations were made on data coded to the next lower full inch and the next lower full pound.

Table 9—Framingham Relative Weight and Weight Changes Since Age 25 in Relation to Incidence of ASHD in Four-Year Follow-Up, Males 45–62

	Population at Risk	New Disease	Rate/1,000
Framingham relative weight:			
All persons	898	52	58
120 or more	81	10	123
113–119	95	10 *	105
100–112	320	16 *	50
Less than 100	397	16	40
Unknown	5	—	—
Weight change since age 25:			
All persons	898	52	58
Increase 30 per cent or more	78	9	115
Increase 20–29 per cent	123	12	98
Increase less than 20 per cent	572	24	42
Unknown	125	7	56

* Two persons with new disease, outside of height range for which relative weights were regularly computed have relative weights computed by extrapolation as follows: (1) 56 inches, 121 lbs., extrapolated FRW, 100; (2) 58 inches, 148 lbs., extrapolated FRW, 116.

measure with the present Framingham relative weight for individuals is high. Increase of weight since age 25 is associated with increased risk of coronary disease ($X^2 = 10.9$, $n = 2$, $p = 0.005$). These data on weight are confirmatory of life insurance studies, but are in contrast with the findings described by Keys [5] who found that only 4.8 per cent of a group of 105 men with "ischemic heart disease" were above a relative weight of 120 using a distribution of relative weight not markedly different than that used here. In the present experience, of 52 men with ASHD, 10 (19 per cent) were in the group with relative weight exceeding 120.

The present experience confirms the widespread belief that obesity is associated with the development of ASHD. The observed incidence rate in men with FRW of 120 or more was about twice that of the entire population and three times that of men with FRW less than 100. However, there is little or no relation of moderate obesity with new ASHD. Since only 10 per cent of the Framingham population falls above the FRW of 120, it follows that these are very obese people. For example, the FRW of 120 for a man 68 inches tall would be 202 pounds.

Serum Cholesterol

A number of studies have shown the association of elevation of serum cholesterol with increased incidence of arteriosclerotic heart disease. The recent, nation-wide Cooperative Lipoprotein Study included data on serum samples supplied by the Framingham study, although those cholesterol determinations were carried out in another laboratory.[8] The data presented here are based on determinations of serum total cholesterol which were made in the Framingham laboratory by the method of Abell, et al.[12] During the period when much of the data were being collected the laboratory standardized its results by exchange of serum samples with the four

Table 10—Serum Total Cholesterol in Relation to Incidence of ASHD in Four-Year Follow-Up, Males 45–62

	Population at Risk	New Disease	Rate/1,000
All persons	898	52	58
Cholesterol measured at Examinations I or II:			
260 mg per cent and over	172	21	122
225–259 mg per cent	265	12	45
Less than 225 mg per cent	445	18	40
Unknown	16	1	*
Cholesterol measured at Examination I: †			
260 mg per cent and over	131	16	122
225–259 mg per cent	188	8	42
Less than 225 mg per cent	334	13	39
Unknown	228	14	61

* Rate not computed for base less than 50.
† Included above.

laboratories participating in the Cooperative Lipoprotein Study.

Cholesterol determinations were not made during the early stages of the program, and as a consequence 228 of the 898 men in the age group under study did not have a value recorded at the time of the first examination. All but 16 of the 228 did, however, have a cholesterol value recorded at their second examination, and that value has been used for classifying those men. As a result, seven of 51 new events occurred in men prior to the recording of a cholesterol value; one man died before the second anniversary of his initial examination and is not represented in the series. In Table 10 the data are presented in two ways: first, based on the first cholesterol value recorded for each man, whether it was measured at Examination I or Examination II; and the second, showing results using only data recorded at Examination I. It will be noted that the same conclusions would be reached using either body of data.

There is an increased risk of ASHD in persons with elevated cholesterol levels ($X^2 = 16.2$, $n = 2$, $p < 0.001$), but no apparent gradient below a level of 260 mg per cent. The division at 260 mg per cent is an arbitrary one and well above the mean level of 225.5 mg per cent found for the entire group of Framingham men in this age range.

The Interaction of High Blood Pressure, Obesity, and Hypercholesteremia with Respect to Risk of ASHD

The data in the preceding section indicated that hypertension, obesity, and hypercholesteremia were each associated with increased risk of ASHD in a group of men 45–62. In this section it will be shown that these attributes appear to make independent but varying contributions to risk, and the joint elevation of two or three is associated with a greatly increased risk.

In this group of men the three attributes are not highly interrelated. As shown in Table 11, the correlation between blood pressure and relative weight

Table 11—Intercorrelations of Blood Pressure, Relative Weight, and Total Cholesterol, Males 45–62

Attributes			Product-Moment Coefficient of Correlation *
Systolic pressure	x	relative weight	0.27
Diastolic pressure	x	relative weight	0.33
Systolic pressure	x	cholesterol	0.09
Diastolic pressure	x	cholesterol	0.12
Relative weight	x	cholesterol	0.06

* $n = 877$, $\sigma_r \cong 0.034$.

is low, and there is almost no correlation in this age group between serum cholesterol level and either blood pressure or relative weight. It is important to consider the independent contribution that each of these attributes makes to the risk of developing ASHD, because each of them is more or less susceptible to individual study and control.

The data in Table 12 show the extent to which high blood pressure, high relative weight, and high serum cholesterol levels are associated with increased risk of coronary disease when elevation of any one is found in a man who has

Table 12—Incidence of ASHD in Four-Year Follow-Up, Males 45–62 Classified According to Level of Blood Pressure, Relative Weight or Cholesterol Level (Effect of Other Variables Partly Controlled)

Attributes *			Population at Risk	New Disease	Rate/1,000
Blood Pressure	Relative Weight	Total Cholesterol			
All persons †			877	51	58
Classified on blood pressure (men with high relative weight or high cholesterol omitted):					
High	Med. or low	Med. or low	91	9	100
Borderline	Med. or low	Med. or low	242	9	37
Normotension	Med. or low	Med. or low	240	4	17
Classified on relative weight (men with high blood pressure or high cholesterol omitted):					
Border. or normo.	High	Med. or low	87	5	57
Border. or normo.	Medium	Med. or low	198	5	25
Border. or normo.	Low	Med. or low	284	8	28
Classified on cholesterol (men with high blood pressure or high relative weight omitted):					
Border. or normo.	Med. or low	High	112	9	80
Border. or normo.	Med. or low	Medium	178	9	51
Border. or normo.	Med. or low	Low	304	4	13

* Classification of attributes:
Blood pressure
High—consistently 160 systolic or over or 100 diastolic or over
Normotension—consistently below 140 systolic and 90 diastolic
Borderline high blood pressure—all other
Relative weight
High—Framingham relative weight 113 or over
Medium—Framingham relative weight 100–112
Low—Framingham relative weight under 100
Total cholesterol
High—260 mg per cent or over
Medium 225–259 mg per cent
Low—under 225 mg per cent

† Excludes 21 persons for (one developing new disease) whom measurements of one or more attributes were not available.

Table 13—Incidence of ASHD in Four-Year Follow-Up, Males 45–62 Classified According to Various Combinations of Blood Pressure, Relative Weight, and Cholesterol Levels

Attributes *			Population at Risk			
Blood Pressure	Relative Weight	Total Cholesterol	No.	Per cent	New Disease	Rate/1,000
All persons †			877	100	51	58
High on two or more			105	12	15	143
High	High	High	17		5	
High	High	Med. or low	47		3	
High	Med. or low	High	20		1	
Border. or normo.	High	High	21		6	
High on one only			290	33	23	79
High	Med. or low	Med. or low	91		9	
Border. or normo.	High	Med. or low	87		5	
Border. or normo.	Med. or low	High	112		9	
Border or medium on two or more			186	21	7	38
Borderline	Medium	Medium	48		4	
Borderline	Medium	Low	63		–	
Borderline	Low	Medium	42		3	
Normotension	Medium	Medium	33		–	
Border or medium on one only			198	23	5	25
Borderline	Low	Low	89		2	
Normotension	Medium	Low	54		1	
Normotension	Low	Medium	55		2	
Normotension or low			98	11	1	10
Normotension	Low	Low				

* See footnote on Table 12 for definition of attributes.

† Excludes 21 persons (one developing new disease) for whom measurements of one or more attributes were not available.

medium or low levels of the other two.* It will be seen that blood pressure level is clearly associated with risk of coronary disease in the absence of elevation of relative weight or cholesterol ($X^2 = 12.1$, $n = 2$, $p = 0.002$). The risk among men with high blood pressure who were in medium or low categories of the other two variables was about six times that of normotensive men. Cholesterol also showed a clear association with risk ($X^2 = 11.6$, $n = 2$, $p = 0.003$), and again the risk among men high in cholesterol was about six times that among men in the low group in the absence of elevation of blood pressure or relative weight. Although there is an indicated excess risk among men with high relative weight, on the data at hand the observed difference is not statistically significant ($X^2 = 2.3$, $n = 2$, $p = 0.32$). Apparently most of the association of risk of coronary disease with obesity is accounted for by the

* For this and the following analysis the level of blood pressure which is termed "high" is different from the definition of "hypertension" used in Table 7. "High blood pressure" is defined here as pressure consistently at or above 160 mm Hg systolic or 100 mm Hg diastolic. Use of this criterion places 20 per cent of the men in the "high" category and thus corresponds to the relative weight criterion of 113 and the serum cholesterol criterion of 260 mg per cent, each of which place 20 per cent of the men in the "high" category.

Table 14—Incidence of ASHD in Four-Year Follow-Up, Males 45–62 Classified According to Blood Pressure and Cholesterol Levels

Attributes *		Population at Risk		New	
Blood Pressure	Total Cholesterol	No.	Per cent	Disease	Rate/1,000
All persons †		877	100	51	58
High on both		37	4.2	6	162
High	High				
High on one only		271	30.9	27	100
High	Medium	51		3	
High	Low	87		9	
Borderline	High	75		10	
Normotension	High	58		5	
Border or medium on both		115	13.1	8	70
Borderline	Medium				
Border or medium on one only		284	32.4	8	28
Borderline	Low	187		6	
Normotension	Medium	97		2	
Normotension or low		170	19.4	2	12
Normotension	Low				

* See footnote on Table 12 for definition of attributes.

† Excludes 21 persons (one developing new disease) for whom measurements of one or more attributes were not available.

association of obesity with high blood pressure.

In Table 13 all three attributes are considered simultaneously. There is an evident association of these groupings with risk of ASHD ($X^2 = 25$, $n = 4$, $p < 0.001$). Consider first the bottom group which includes the 11 per cent of the men 45–62 who were characterized by low values in all three variables, i.e., blood pressure less than 140/90, FRW less than 100, and cholesterol less than 225 mg per cent. The incidence rate for this group was 10 per 1,000, a rate even lower than that observed among all women of the same age (29 per 1,000). The presence of borderline or medium levels of one or more of the three attributes is associated with progressively increased risk. Definite elevation of any one of the three is associated with a markedly increased risk—79 per 1,000. The concomitant elevation of two or more variables is associated with an indicated four-year incidence of 143 per 1,000. It is thus possible to contrast a small group (one-eighth of the men) who have an indicated risk of one in nine of developing recognizable ASHD in four years with a similarly sized group at the opposite end of the scale who had an indicated risk of one in 100. In view of the small number of new events on which this analysis is based it is perhaps more appropriate to compare the experience of the men in the two upper groups with that of the men in the two bottom groups. The two upper groups, which include the 45 per cent of the men high on any one of the three attributes, have an incidence rate of 96 per 1,000 and include 75 per cent of the men who developed disease. The two bottom groups which include 34 per cent of the men have an incidence rate of only 20 per

1,000 and include only 12 per cent of the men who developed disease. Of particular interest are the 17 men who were high in all three attributes. In this group there were five new occurrences—odds of five to 12 to lose in four years.

In view of the suggestion that the effect of obesity on risk may be through its relation with hypertension data are presented in Table 14 classified on blood pressure and cholesterol only. It is possible using only these two variables to separate out a group high on one or the other of the two which includes 35 per cent of the men and two-thirds of the new disease (33 out of 51). The four-year incidence in this group was 107 per 1,000. This group may be contrasted with a group of men who were low on both, or medium or borderline on only one, which included over half of the population (52 per cent), but only 20 per cent of the new disease (10 out of 51). The four-year incidence in this "low" group was 22 per 1,000—only one-fifth of the incidence found in the high group.

Education

The formal educational experience of the subjects in the Framingham study may be used as an index of the relationship of socioeconomic status to the risk of developing ASHD (Table 15). Although the incidence rate of 70 per 1,000 for persons with no more than grade school education is slightly higher than that of the base population, it does not prove to be significantly higher, and the absence of a trend among the other categories gives no suggestion of an important association. It is of interest that men with high school, college, business, or graduate school training showed rates no greater than those with less education. There is very little evidence in these data to suggest a relationship of socioeconomic status with the appearance of ASHD, although the classification by education may not be a sufficient index of this variable. Since the Framingham population contains an unusually broad array of occupations, this attribute will be studied in greater detail. Morris [13] also found but little relationship between educational accomplishment and the risk of coronary disease in the British occupational mortality experience.

Smoking

It has been proposed that smoking is associated in varying degrees with the development of ASHD.[14, 15] The data in Table 16 illustrate certain interpreta-

Table 15—Education in Relation to Incidence of ASHD in Four-Year Follow-Up, Males 45–62

Educational Status	Population at Risk	New Disease	Rate/1,000
All persons	898	52	58
Grade school only, or none	386	27	70
High school, not graduated	139	6	43
High school graduate *	182	9	49
College	158	8	51
Unknown	33	2	†

* Includes "business college" and specialized trade school subsequent to high school graduation.
† Rate not computed for base less than 50.

Table 16—Smoking Pattern Recorded at Initial Examination and Incidence of ASHD in Four-Year Follow-Up, Males 45–62

Smoking Pattern	Population at Risk	New Disease	Rate/1,000	
All persons	898	52	58	
Never smoked	107	9	84	70
Lapsed	78	4	51	
Cigar or pipe only	131	5	38	36
Cigarettes per day:				
Less than 10	91	3	33	
10–15	76	7	92	
20	204	14	69	72
25–35	79	3	38	
40 or more	55	6	109	
Unknown	77	1	13	

tive difficulties in the present body of data. The variability of attack rates is large and without a regular progression among men with different smoking habits. Any attempt to consolidate persons into broader categories of smoking habits in order to gain the advantage of larger numbers has major effects upon the resultant incidence rates. Nevertheless, very heavy smokers showed high incidence and the light cigarette smokers and pipe and cigar smokers showed lower incidence of ASHD. But persons who had never smoked, or had quit smoking, showed a rate as high as the entire group who reported smoking 10 or more cigarettes per day. Doll and Hill,[16] in their second report on mortality among British doctors, showed an upward trend of mortality from coronary thrombosis in relation to smoking in men who were either under 55 years or 75 and over, but very little trend in the age range 55–74. Generalizations cannot be made until additional morbidity and mortality experience become available from the present study, but these data do suggest that the association of ASHD with smoking is not as strong as that shown with elevated blood pressure, relative weight, and cholesterol.

Discussion

This study has identified three readily measured characteristics of human subjects which are frequently present in advance of the development of definite ASHD. These are high blood pressure, overweight, and hypercholesteremia. Each of these appears to have association with the development of the disease; the group of individuals in which all three are coincidentally high show great increases of incidence of ASHD. High blood pressure and hypercholesteremia in this group clearly have some independent association with risk of disease. The role of obesity, which is also associated with heart disease, is not as clear in view of the demonstrated association of high blood pressure and obesity. The demonstration of the association of these clinical attributes with ASHD should encourage the search for common factors and explanatory mecha-

nisms. This search should be a prime function of epidemiological studies of heart disease.

The extent that these findings can be used for generalization about the biology of ASHD is limited by the small experience with de novo disease, the possible bias introduced by the persons drawn in the sample who either refused original examination or were soon lost to follow-up, and finally by the imprecision of the diagnostic procedures. The first limitation will ultimately be met with additional follow-up. The principal evidence concerning the question of sample refusal is supplied by comparison of the mortality experience of the original participants and the nonparticipants. While mortality of the latter was excessive for a few years it is now reassuringly similar to that of the participants, and it does not appear to be a source of significant bias. The diagnosis of ASHD is extraordinarily difficult. These data have demonstrated that almost a third of subjects experiencing "heart attacks" die suddenly so that there is little or no opportunity to apply clinical and laboratory studies for the identification of the kind of antecedent disease. Another group, perhaps a fifth, have either no symptoms or trivial symptoms of heart disease, and yet show definitive evidence of new coronary artery disease in routinely obtained electrocardiograms. This difficulty of diagnosis compounds the uncertainty of studying atherogenesis by means of the clinical manifestation of ASHD. Nevertheless, since this clinical approach will probably remain the only feasible one, it is of first importance that some uniformity of criteria for the grouping of the clinical manifestation of ASHD be adapted in epidemiological studies.

Summary

1. An evaluation has been made of the prevalence of arteriosclerotic heart disease (ASHD) in Framingham, Mass., and the incidence in persons presumably free of ASHD in four years of follow-up.

2. The initial examination included 68.6 per cent of a randomly selected cross-section of the population aged 30–59 in 1950. Subsequent biennial examinations have obtained clinical information on 89.1 per cent of the initially examined population. Mortality information is available for 99 per cent of this population.

3. A working classification of ASHD has been adopted which includes myocardial infarction, coronary occlusion (sudden death attributed to coronary heart disease), angina pectoris, myocardial fibrosis, and possible myocardial infarction diagnosed by electrocardiographic evidence only.

4. ASHD was about twice as prevalent in men as in women at the first examination, and in men was comprised about equally of AP and MI. ASHD was uncommon in either sex under age 40.

5. There were 97 persons free of definite ASHD at the first examination who developed the disease in the subsequent four years of observation. The incidence rate for men was twice that for women.

6. All this new disease occurred after age 45 in women and 80 per cent after this age in men.

7. Almost half the men developing heart attacks died, and almost one-third of the men died suddenly.

8. About one-fifth of the new myocardial infarctions were asymptomatic.

9. The ASHD appearing in women was predominantly angina pectoris.

10. Among men 45–62 the presence of high blood pressure and HCVD was strongly associated with the development of new ASHD.

11. A reference table of weight for height was established for males and it was shown that fatness is associated with the development of ASHD in men 45–62.

Much of this association appeared to be explained by the association of obesity with high blood pressure.

12. Hypercholesteremia was strongly associated with the development of new ASHD in men 45–62.

13. The use of three factors in combination—blood pressure, relative weight, and serum cholesterol permitted the separation of men 45–62 into groups with highly divergent risks of ASHD. Classification on blood pressure and cholesterol without regard to relative weight also made possible separation into groups with large differences in risk.

14. Neither smoking habits nor educational background were notably associated with the development of new ASHD.

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